CONGENITAL LARYNGEAL STRIDOR.

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Congenital laryngeal stridor has been the subject of many articles, mostly of European source, and of much discussion; the opinions as to its cause are almost as various as the number of observers. It was first described by Rilliet and Barthez, in 1853. In 1877, Krishaber reported 4 cases which existed from birth. The English writers were the earliest to trace its pathology and make the picture complete as it is accepted today. Lees, in 1883, was the first to report an autopsy and show with a plate his finding. In 1884 Gee reported 12 cases of noisy respiration, but the clinical picture given is not always that of a typical case. Politzer in the same year saw a case which he thought arose from vagus irritation, because of noise; he also thought the expiration divided into five or six staccato parts. The absence of dyspnea puzzled him greatly. He gave origin to the nervous theory which has largely obtained to the present time. Then followed cases by Goodhart, Lori, Suckling, Robertson, Smith, Semon, McBride, Variot, Refslund, Sutherland and Lack, Avellis, Herzfeld, Moure, Guinon, Comby, Thomson and Logan Turner, and others. An observation of my own is as follows:

A boy, three weeks old, was seen by me, July 10, 1903, because of a peculiar crowing sound made on inspiration. This noisy breathing was continuous, and accompanied at times by marked cyanosis. It began shortly after birth and went on without any free intervals, causing great distress and much loss of sleep to the parents. To all appearances the child was healthy, with the exception of the efforts made by it to obtain air—which were shown by the inspiratory depression of the jugulum and supraclavicular and infraclavicular fossæ, and also by the marked action of the chest. There was no sign of respiratory arrhythmia. The noise was of a crowing, guttural character, and could be heard through two rooms. The cry was normal. He is the fifth child. The family history is excellent. All the other children are healthy.

It was difficult, without an anesthetic, to examine the child with a mirror, owing to the smallness of the parts. Ether was administered, a mouth gag inserted, and a suture put through the tongue which rendered it easy to insert the mirror and see the parts. The epiglottis showed its lateral edges much incurvated, or rather rolled on themselves, so that the lateral edges and the aryepiglottic folds approximated very closely. A small rhomboidal opening could be seen at the base of the epiglottis, which was bounded by part of the lateral borders of the epiglottis and the anterior part of the aryepiglottic folds. It was impossible to see the vocal cords because of the overlying position of the epiglottis on this rhomboidal opening. No external enlargement about the neck or of the jugulum, which would suggest enlarged thymus or thyroid, could be observed.

When about three months old the cyanotic attacks began to disappear, and chest deformity of a marked character became a prominent feature. The costosternal articulations became prominent, and the muscles of the chest somewhat over-developed, giving the appearance of a strong full chest. The marked action of the chest was also noted, and efforts made in inspiration still continued, as shown by the depression in the jugulum and the epigastrium. The sound still continued uninterrupted. When the child was a year old there was apparently little change, except in the tone of the sound, which had become rougher and of a deeper quality. The child looked extremely healthy. Once or twice it had slight colds which lasted for a few days but had no effect on the sound or its character.

When eighteen months old, the child was again examined; the action of the chest was less marked, but still gave the impression of extra effort. The sound was much less marked except on exertion, when its old character would return, but the breathing was always accompanied by a slight stridor. It was now observed that when the child fixed its attention the stridor entirely disappeared, but only for a moment. It was also observed, at this time, that the child's lower jaw did not occlude properly, there being a recession of fully half a centimeter. The child, however, did not breathe through the mouth. When two years of age, the child was examined with a mirror; the epiglottis still retained its peculiar shape, but the interior of the larynx could be seen well. The vocal cords acted normally the tendency of the aryepiglottic folds to approximate was still present. The recession of the jaw was more marked, and the muscles connected with it appeared tense. A slight sound still persisted, but under excitement it would return with some of its old character.

Etiology. Many theories have been advanced as to the cause of this stridor. The first is that of Rilliet and Barthez, that it is due to an inflammation or compression of the trachea, but only one of their cases approaches the real picture of the disease. Lori, of Buda-Pest, followed with a unique theory of clonic spasm of the glottis. He based this opinion on a laryngoscopic view, wherein he claims to have observed the cords pressing against one another. Robertson also favored the nervous origin of the trouble, and thought that adenoids set up a reflex disturbance affecting the abductors. Thomson and Logan Turner favor the nervous theory. They state that the spasm lasts a second or two, and accompanies almost all of the child's inspirations. Sutherland and Lack state that it cannot be definitely decided that the vocal cords play a part, for in some cases there is not an abductor paralysis. Further, it was seen in older patients that the cords moved well and were fully abducted. Yet in these cases stridor occurred on exertion or during a quick inspiration and was invariably associated with a falling inward of the ary-epiglottic folds. The nervous theory receives a fatal blow in that the affection persists for months, even during sleep and anesthesia.

Thomson and Turner believe that it is due to ill coördinated spasmodic action of the muscles of respiration, that is, a choreiform respiratory spasm analogous to stammering. They show that the respiration of all young infants is peculiar, in being irregular in rhythm and force, as if the coördination involved in its movements was not under control. These often occur with sudden respiratory efforts, as for example, when a young baby is excited it is likely to make a crowing noise with inspiration, although it does not do it at other times. When coming out of chloroform anesthesia a similar stridor is observed, and a very loud crowing noise is a common accompaniment of operations on the genital organs, due to sudden inspirations which stimulation of nerves of these parts is likely to occasion. Along with the crowing there is also an indrawing of the lower chest wall proportionate to the loudness of the sound. Thomson and Turner conclude that there is a backward development of cortical structures of the brain, which in turn allows incoördination of respiratory movements. They claim that the deformity of the epiglottis is merely an exaggeration of the infantile type and due mainly, if not entirely, to suction of the upper aperture produced by the incoordinated respiratory movements, or, as they call it, stammering respiration. They claim there is no proof that congenital malformation of the larynx exists in these cases. Gay's statement on laryngismus stridulus, to the effect that this is caused by central irritation in children "or better to a deficiency of the higher centres," supports their theory. They hold it is analogous to other coördination neuroses, such as "writer's cramp." Sutherland and Lack oppose this theory of the developmental deficiency of the cortex, their observation showing that the children affected with stridor respire in the same manner as healthy children, that is, with the coördinated and spasmodic type. They also oppose the view that the crowing noise of chloroform anesthesia is due to the same cause as the stridor, because this occurs in adults under similar circumstances.

The adenoid theory was held by many. E. Smith reported 2 cases. The first snored from birth, but this disappeared on operation. The second, which was probably a genuine case, persisted for twelve days after the operation and returned at the end of a year when the child took cold. McBride does not think these cases belong to the genuine stridor group. E. Clarke operated on a patient but the stridor persisted. Variot had a similar experience.

It has been held by some that the sound is produced by tracheal stenosis, caused by enlarged thymus or by enlarged mediastinal glands. The thymus theory was strongly advocated by Avellis, who states that Allen Burns early in the last century thought the thymus a cause of tracheal stenosis and even suggested an operation for it. Siegel had a case which he erroneously diagnosticated as diphtheria. No result followed tracheotomy until the short tube was removed and the long one inserted. The latter caused decubitus. It was then realized that the thymus might be the cause, and Rehn proceeded to carry out Burn's suggestion and opened the mediastinum, lifted the thymus upward, and stitched it to the fascia of the neck. The trouble disappeared. F. Koenig had a like experience by partially removing the thymus.

Avellis speaks of Glockle's observation of 3 cases of stridor which terminated fatally as the result of enlarged thymus. Weigert could demonstrate the compression of the trachea in 1 of these cases. Avellis urges in favor of the thymus theory: (1) The age of the child; (2) cure in the second year; (3) congenital appearance; (4) increased by movement; (5) periodic stopping of the stridor when the position was changed; (6) negative laryngeal examination; (7) high position of the larynx; (8) air current in one bronchus stronger than in the other; and (9) operative results.

Stamm believes the thymus a cause. Hochsinger, of late, has investigated this with the *x*-rays, but his pictures were not looked upon with favor by Ganghofner, under whose suggestion he worked. Siegert also said that often when a large thymus existed he did not observe any stridor and related a case in which enlarged thymus existed without stridor. Thiemich suggests starving the child to produce shrinkage of the thymus, whereupon the child would lose the stridor were this the cause. This treatment has not been followed. Feer, of Basel, observed a case of enlarged thymus with stridor. Hochsinger states that enlarged thymus can exist without stridor.

Tatschner, in an article in answer to Hochsinger, refutes his statements. The patient was his own child, and he showed that by pulling the chin forward the sound disappeared. Whenever he held the child's attention for a minute either by calling it to pronounce words or checking its voluntary activity stridor stopped. It would invariably return, however, when he resumed his play.

As can be seen from the foregoing, the thymus theory is not sufficient, because: (1) The will has no power over the turgescence of an organ controlled wholly by an involuntary innervation. (2) Hochsinger himself admits that in many of his cases, in which he determined excessive hypertrophy, no stridor existed. (3) The *x*-ray picture was proved but once by autopsy. (4) Pulling the chin forward affects the position of the thymus gland, thereby increasing the compression which produces stridor. (5) The stridor observed in compression of the trachea is not inspiratory alone, but is both inspiratory and expiratory.

The case used by Avellis to support his theory, the case of Siegel and Rehn, had sudden attacks of dyspnœa night and day, and was diagnosticated as laryngismus stridulus by M. Schmidt. No specific stridor is mentioned, such as one finds in true congenital stridor. This case was in my opinion a true case of thymus hyperplasia. The child was two and one-half years old when seen, had no history, but began to cough when four or five weeks old. Avellis quotes Schmidt as seeing a patient who "snapped the air," but this has never been observed in true stridor. The attacks of coughing producing cyanosis and suffocating signs are seen in congenital stridor, but the hindrance offered by the malformation of the larynx to the expulsion of mucus sufficiently explains it.

Geffrier brought forth the theory that it was due to enlargement of the tracheobronchial glands. He claimed these could be enlarged at birth and give rise to symptoms which were analogous to those of stridor. This, however, errs in many respects; the noise is both inspiratory and expiratory, cough is similar to that of whooping-cough or measles, and dyspnœa is marked. Then, too, suffocatory crises occur as often as four or five times a day. This enlargement of the glands in children is exceedingly rare and presents a decidedly different aspect from congenital stridor.

PATHOLOGICAL ANATOMY. Lees was the first to show by autopsy the malformation which causes this conditon, and he gives a drawing of the specimen. The patient was a year old when autopsied. It was found that the lateral halves of the epiglottis was so incurvated as to touch each other with their posterior surfaces and almost overlap. The aryepiglottic folds were in close proximity to one another. They were thin and not edematous, the latter fact being important because the child died of diphtheria. There was found between the base of the epiglottis and the approximation of the aryepiglottic folds a *rhomboidal opening allowing the passage of air.* Lees also mentions a pinhole opening on the superior surface of the epiglottis which allowed some air to enter between the incurved folds of the epiglottis, forming a sort of tube.

Variot, in 1898, autopsied a child twenty-two months old, who died of scarlet fever. He found the epiglottis elongated and the edges approaching each other, and the fibrocartilage thin and supple and of healthy appearance. The aryepiglottic folds were approximated and had to be separated to produce the opening which should exist. This larynx was dissected by Chauveau, who found the arytenoid cartilages longer than usual. The thyroid and cricoid cartilages were normal. It was in this case that he tried intubation and found that the phlange of the tube kept the aryepiglottic folds apart, thus causing the stridor to disappear. Sutherland and Lack confirm with autopsy the above findings. Refslund performed an autopsy on a boy three and one-half months and gives a plate which is similar to those described. The measurements of the parts were:

the epiglottis from base of the tongue to the superior surface 5 mm., from the furrow on the anterior surface to the lateral border 5 mm., or 10 mm. across and 1.5 mm. thick. The furrow was much thinner and transparent than the lateral parts. In this furrow was embedded the connective tissue which forms the median glossoepiglottic ligament coming from the tongue. Owing to the weakness at this point there was great flexibility, hence incurvature was made easier. There was also greater mobility in the epiglottis and the separate halves could be made to overlap easily. Between the arytenoids was a fissure 7 mm. long, formed by the aryepiglottic folds whose edges were close together; from their anterior point of contact to the base of the epiglottis was a separation of 3 mm. Otherwise the larynx was normal.

Thomson and Turner dissected fifty young subjects, some being to the tenth year of life. They found the structures about the vestibule much softer and more collapsible than in the adult, the aperture relatively narrower laterally, owing to the folding backward and inward of the epiglottis and the consequent approximation of the aryepiglottic folds. As the child grows the epiglottis loses its infantile shape and the aryepiglottic folds separate proportionately. They inserted a tube upward in the trachea between the vocal cords and then simulating inspiration, produced the effect of approximating the aryepiglottic folds so as to form a narrow mesial slit. They found that the aryepiglottic folds approximate in infants. They also hold that the peculiar form partially persists up to the seventh year. In this Sutherland and Lack agree.

SYMPTOMATOLOGY. The sound has been variously described by the mothers as being similar to "sound produced by hens," "like a chicken," "like a grunter," "purring like a kitten," and "like croup." Dr. Thomson in his able analysis says, "It begins with a croaking noise and ends with a high-pitched note, and expiration is accompanied by a slight croak when the noise is the loudest. It varies in pitch a great deal. In some cases the note is deep, purring or growling, and in others high and squeaking." The latter holds true mostly in the beginning of the disease, the croaking sound as the child grows older becoming more and more prominent. The loudness varies at times and in almost all of the cases there are short periods of intermission occurring at such times as when the breathing is superficial and regular, or when the child is able to notice by having attention fixed, or when asked to speak. If the child be taken from a cold to a warm room, or when the child is asleep, it diminishes. It increases in loudness either on awakening from a sleep, or on being bathed, or if it is fed too quickly. It can be heard through several rooms of the house; in some instances the observers were attracted by the noisy respiration of the child in the waiting-room of their offices. It appears to be louder when the child is most healthy. When noisiest, it is not continuous, for single normal breaths can be observed. Pauses occur sometimes lasting an hour. They may come without interruption from the noisiest breath and come back also the same way. If often becomes common snoring. It generally has the same timbre and tone-height. Several observers have had the feeling that it was produced by a floating membrane.

There is no difference noted in the sound with the mouth open or shut, or from position. Tatschner observed that on pulling the chin forward it would disappear. In my case pulling the tongue forward under ether did not cause it to disappear.

The stridor increases in loudness for some weeks or months after birth and then continues more or less stationary until the eighth or ninth month. Then it begins to diminish until the eighteenth month. It may last up to the end of the third year. Sometimes it intermits for a month. Even after it has disappeared for a considerable period, if the child takes cold or is out of breath it returns. Occasionally during the third year a short crow or croak is heard. Suckling thought the sound was more intense in sleep and during cry. Lori said in his cases it disappeared for hours between the third and sixth months. He and Refslund observed that it disappeared when the head was depressed or turned to one side; other observers, however, do not bear out this statement.

SIGNS OF OBSTRUCTION. There may be cyanosis at times, but often it is absent. This may be produced as a result of coughing, because of the impediment offered by the deformed epiglottis in expelling secretion. Sutherland and Lack found that in 8 of their cases suffocatory attacks came on when the child was asleep, sometimes when awake. In 1 fatal case observed by them the cyanosis was persistent. When intense the jugulum was tumefied.

The respiratory movements are of a peculiar nature, and at first strongly suggest diphtheria. Most observers have reported increased action of the alæ, marked retraction of the episternal notch, supraclavicular fossæ, and epigastrium, and also some retraction along the ribs, in the axillary region especially. As a result of this, the chest shows gradual prominence of the sternum and the costocartilaginous synostoses, giving in effect a rachitic shape. This has occasioned many errors as to the etiology and diagnosis of the affection. It was thought by more than one observer to be true laryngismus stridulus, this being a frequent concomitant of rickets. This however, can be easily excluded, owing to the absence of the signs, craniotabes, and thickening of the epiphyseal ends. The majority of observers have noted rales all over the lungs, which may be due to the transmission of the laryngeal sound. Thomson states that the lungs are not fully expanded and that breath sounds become feeble toward the bases. This may be disproved because of the intensified action of the respiratory muscles and the absence of signs which would show this condition. Rales and rhonchi are common all over the chest and not in special locations. Postmortem reports do not confirm this, as will be shown under the pathology of the affection.

Variot says that auscultation is of no value, because of noise. Pronounced participation of the abdominal muscles in the respiratory action has been observed. Thomson notes strong action of the sternocleidomastoid muscles. Deformity of the chest persists. Refslund observed movements of the larynx in respiration.

The retraction of the jaw mentioned above in the history of the case is an important factor. It was late in appearance and not observed until the child was over a year old. The retraction caused an occlusion of the teeth of about 0.5 cm. The mouth had a peculiar appearance, yet the child was not a mouth breather. There appears no mention of this symptom in any of the articles published on congenital stridor or on diseases of the thymus, except in one reported by Tollemer, in which stridor ascribed to congenital cardiac malformation existed. He mentions having observed retraction of the jaw of 0.5 cm. His report states that the child was a blue baby and that he worked two hours before the respiration was satisfactory. The presence of this symptom is explained by the action of the muscles attached to the larynx, chin, and hyoid bone, the hyoglossus, geniohyoglossus, digastric, geniohyoid, and omohyoid muscles.

The epiglottis has no special musculature. Luschka states that a small part of the genioglossus muscle is inserted into the ligamentum glosso-epiglotticus medius which has an elevator action of slight development and little importance. These muscles are brought into action when the larynx has a high position, in order to facilitate the separation of the aryepiglottic folds forming the obstruction.

Complications. Most authors insist on the frequency of various pulmonary troubles. Martin observed tracheobronchitis of great tenacity in 1 of his cases. Another child died of bronchopneumonia. Tarny mentions a whooping-cough attack when the child was eleven months old. Guilbert states that bronchitis is frequent, and that one of his patients often had head colds. Rocaz reports a tracheobronchitis in 1 case, in the other 2, bronchopneumonia. One of the latter succumbed. Chauveau's case had bronchopneumonia at eighteen months. In 2 of Thomson's cases dyspepsia was a factor, in a third case the child vomited often and brought up phlegm, in a fourth the child was dyspeptic because the mother did not have sufficient milk, and in the fifth bronchitis was noted. Variot mentions bronchitis in 1, and in another scarlet fever which proved fatal.

Sutherland and Lack had 1 case in which convulsions occurred on the eighth day and recurred up to the second month. One was noted as anemic. Several had bronchitis and 8 had suffocatory attacks. These generally appear within the first two months and are serious. The apnœic condition caused by this peculiar malformation is undoubtedly more frequent than we think. It is not uncommon to hear of newborn children who apparently breathed only with great difficulty at long intervals, sometimes as long as three minutes between each inspiration, accompanied by crowing noise. After a half hour the child would die. Often cases appear in which it is difficult to keep up the respiratory function unless the child is stimulated in various ways, as was observed by Miller, Burral, and others. In these the stridorous breathing continues, but apparently with such effort that the child becomes easily exhausted and dies in a convulsion.

In the first two months of the disease attacks of cyanosis are frequent, but they rarely become so marked as to occasion fear of death. The dyspeptic conditions observed by Thomson and Sutherland, and Lack would seem coincidental.

Suffocatory attacks occur after the first week of the disease, but are of short duration, except in the case of Sutherland and Lack in which convulsions from this source came on every day and lasted two months. Rickets has been mentioned as a common complication. This, however, seems questionable, the error caused by the peculiar conformation of the chest being due to the increased action of the respiratory muscles.

Differential Diagnosis. At first sight laryngismus stridulus appears to be laryngeal diphtheria, as shown by the retraction symptoms and the noise, but after studying the child a little this can be excluded on the following grounds: (1) There is little or no cyanosis; (2) there are no exaggerated efforts to get air as in diphtheria; (3) as a rule, the history of its early origin is within the first week; and (4) there are no diphtheria bacilli.

Adenoids and enlarged tonsils produce a snoring noise which disappears on closing the alæ. In the cases observed only Clarke and Smith, who held this to be a cause, found adenoids or tonsils.

Diaphragm of the glottis is very rare. Fein could find but 13 good cases and 4 doubtful ones. The symptoms are entirely different in character, the voice being hoarse and dyspnœa observed. Most of the cases occurred later in life, from the seventh to the sixty-fifth year.

Foreign bodies in the larynx must be considered, but here the sudden dyspnœa, convulsive cough, and the phonatory troubles are variable. The history is also valuable in excluding this cause. Semon suggests paralysis of the dilators of the glottis. In this affection the voice is normal, but the dyspnœa intense. The patient's age is also against it.

Tumors of the larynx must be considered. Retropharyngeal abscess is frequent in children but not at birth. This can be excluded by the presence of pain and by digital examination for the swelling. The general condition is bad, as a rule, the temperature is elevated, and the child refuses nourishment.

Hyperplasia of the thymus and enlarged tracheobronchial glands have already been discussed. In such early cases as Miller's, one must consider atelectasis of the lungs, as found in the case he autopsied. He reports one-third of the lung expanded and two-thirds non-crepitant. He also noted that the epiglottis was folded.

Rilliet and Barthez mention atelectasis of one lobe of the lung in a case in which dyspnœa had continued from birth until death at two and one-half months, which was thought at first to be due to stenosis of the trachea.

PROGNOSIS. Sutherland and Lack claim laryngeal stridor to be of little hindrance to growth, except in delicate children. It is bad if pulmonary affections occur. There are in all 101 unquestionable cases reported; death resulted in 21. Lees' patient died of diphtheria, aged thirteen months. Of Refslund's 3 patients, 1 died at six weeks of age, and 1 at nine months, cause not given; the third patient had left lobar pneumonia. Variot's patient died of scarlet fever, aged twenty-two months. Of Sutherland and Lack's 3 patient's, 1 died at the age of two weeks, of convulsions after labored respirations and marked cyanosis, but nothing was found at the autopsy; the second, twenty-five months old, died of "pulmonary disease following a persistent bronchitis" of eight months' duration; the third, an infant of ninteen months, died of tuberculosis. Greene relates the case of a child one year old that died of pneumonia. Both Martin and Rocaz report infants dying of bronchopneumonia at twenty months. Ballin had 3 cases of pulmonary disease, in 2 of which death ensued, 1 a girl two and one-half months old, died of bronchopneumonia (confirmed bv autopsy); the second, a boy four months old, had capillary bronchitis (also confirmed by autopsy); the third case, a boy thirteen months old, had pneumonia when nine months old and recovered. Massei reports a boy who died of pneumonia when five months old. Cozzolino observed a boy seven months old with double pneumonia whose stridor disappeared before death.

Miller's 3 cases are especially interesting since death ensued in all within the first twenty-four hours of life. No cry was observed in any of them at birth, and attempts to inspire were irregular and short, accompanied by crowing noise. At times breathing would stop and they became cyanosed. He thought it a spasm, and gave inhalations of ether without effect. Artificial respiration and slapping the back would bring color to the skin. He agrees with Tatschner that the finger behind the jaw would permit respiration to go on easily. The child died apparently of suffocation nineteen hours after birth. On the mother's side, according to her statements, several children died in a like manner a few hours after birth. Burral's patient also died of convulsions two weeks after birth. Levy and Etienne report a boy of seventeen months, who died of œdema of the larynx. Schmiegelow's case is probably one of stridor, with report of a specimen, his pathological diagnosis being "stenosis of the larynx." He gives a good description of the symptoms which are not dissimilar to this affection.

From the foregoing it is to be deduced that death is usually due, directly or indirectly, to the effects of the stenosis. In 7 cases death seemed directly due to the stenosis. After labored breathing for a period varying from a few hours to two weeks the child would become cyanosed and die in convulsions. The next most prevalent cause of death was bronchopneumonia, in 5 cases. There were 3 of lobar pneumonia, and 1 each of tuberculosis, œdema of glottis, diphtheria, and scarlet fever.

Treatment. Tracheotomy and intubation have been tried to alleviate this condition. Variot used the former, but the child failed to retain the tube. However, one important point was gained in that the sound disappeared while the tube was *in situ.* This, as Variot demonstrated postmortem, was produced by the phlange of the tube separating the arytenoids and holding the aryepiglottic folds apart. He advises cutting away the aryepiglottic folds. Ballin did a tracheotomy on one of his patients and the stridor disappeared, but the child died thirty-six hours later of bronchopneumonia.

I think Variot's suggestion to cut away the aryepiglottic folds a good one and should be tried, but in my cases I saw no urgent need of interference and kept the child under observation.

Cutting away the aryepiglottic folds is not a difficult procedure at the present time, owing to recent advances made in endolaryngeal operations by the direct method, especially on children. It is very difficult to operate on a child by the indirect method, because of the smallness of the parts. The introduction of Kirstein's autoscope has rendered the larynx in children very accessible for the cutting instruments. The use of either the Grunwald or the Hajek double curette for working on the sides of the larynx would render the removal of the aryepiglottic folds a very simple matter. The removal of the epiglottis would require a stronger instrument, such as a small-sized tonsil punch. We know that the removal of the epiglottis is compensated for, and no danger can arise because of its removal. The cutting away of the epiglottic folds might be tried first. If this did not give sufficient relief, a fact which could be observed during the operation, the removal of the incurved part of the epiglottis probably would. I do not hesitate to advise this, because there is a minimum of risk.

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WHEN SHALL WE ADVISE TYMPANOMASTOID EXENTERATION IN THE TREATMENT OF SUPPURATIVE OTITIS MEDIA, AND IN WHAT PERCENTAGE OF CASES MAY WE EXPECT A CURE?[1]

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During recent years the prognosis of suppurative middle-ear disease, including all of its complications and eventuations, has very materially improved. A better realization of the importance of the disease has led to its earlier and more persistent treatment, and antiseptic surgery has enabled the aurist to save the lives of many persons whose conditions a few years since would have been looked upon as hopeless. Of perhaps more importance is the fact that surgical improvements have put it in the power of the aurist to prevent many of the serious complications of this disease.

A purulent inflammation of the middle ear may persist for many years without causing serious disturbance of health or destruction

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[1] Read at the meeting of the Southern Section of the American Laryngological, Rhinological, and Otological Society, at Louisville, Kentucky, February 25, 1907.